Community Threat, Positive Parenting, and Accelerated Epigenetic Aging: Longitudinal Links from Childhood to Adolescence

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Abstract

Early Life Adversity (ELA) has been linked to accelerated epigenetic aging. While positive parenting is hypothesized to buffer the detrimental effects of ELA on child development, its role in mitigating epigenetic age acceleration remains unclear. Data from 2,039 children (49.7% female) in the Future of Families and Child Wellbeing Study (FFCWS) were included in the current study (46.7% Black, 26.5% Hispanic, 19% White non-Hispanic). Home and community threat and observed parenting were measured from ages 3 to 9. Salivary epigenetic age acceleration was measured at ages 9 and 15. Positive parenting reduces the pace of epigenetic aging in low, but not high, community-threat environments. Interventions across home and community environments may be necessary to prevent ELA's biological embedding.

Keywords:

Adversity; parenting; epigenetic aging; childhood; adolescence

Early Life Adversity and Adolescent Epigenetic Age Acceleration: The Moderating Role of Positive Parenting

Early life adversity (ELA) encompasses a wide array of challenging environmental experiences during childhood, including exposure to violence, abuse, neglect, and chronic poverty (Berman et al., 2022; Madigan et al., 2024). These experiences often diverge significantly from typical developmental environments, demanding substantial adaptive responses from affected children (Humphreys & Zeanah, 2015; Merrill, Konwar, et al., 2024; Nelson III & Gabard-Durnam, 2020). A recent meta-analysis (Madigan et al., 2023) revealed that approximately half of the adult population in the United States (US) report experiencing at least one adverse childhood experience, with 16% reporting four or more. Additionally, research has consistently demonstrated that having a history of ELA is closely linked to multiple mental (Hayward et al., 2020; LeMoult et al., 2020) and physical (Grummitt et al., 2021) health problems, highlighting the urgent need for further investigation into factors that mitigate the negative outcomes associated with ELA.

Early Life Adversity and Epigenetic Aging

ELA is associated with poor long-term health outcomes partly due to the physiological stress it induces, which can become biologically embedded (Krause et al., 2020). Biological embedding refers to environmentally induced alterations in physiological systems that may result in enduring biological changes (Aristizabal et al., 2020). DNA methylation, a key mechanism of biological embedding (Aristizabal et al., 2020; Boyce & Kobor, 2015), involves the addition of a methyl group to DNA, typically at a cytosine base, followed by a guanine base (CpGs). This epigenetic modification can influence gene expression without altering the underlying DNA sequence (Aristizabal et al., 2020; Bird, 2002). Crucial for processes like cell differentiation and

development, DNA methylation patterns also shift predictably with cellular aging, thereby altering cellular behavior and responses to environmental stimuli throughout life (Horvath, 2013)

Researchers can estimate an individual's biological or cellular age by analyzing specific DNA methylation sites across the genome, providing valuable insights into how early environmental exposures influence biological processes (Horvath, 2013). Epigenetic age acceleration occurs when biological age, as estimated with DNA methylation, differs from chronological age with increased acceleration indicating advanced biological aging relative to one's actual age (Horvath, 2013). The Pediatric Buccal Epigenetic Clock (PedBE) is a specialized tool designed to estimate DNA methylation age in children's oral samples (Fang et al., 2023). Similarly, DunedinPACE measures the pace of epigenetic aging by assessing longitudinal changes across seven organ systems—cardiovascular, metabolic, renal, hepatic, immune, dental, and pulmonary (Belsky et al., 2022). Studies using tools like DunedinPACE and PedBE reveal that children exposed to ELA often exhibit increased epigenetic age acceleration (Chang et al., 2024; Dammering et al., 2021; Del Toro et al., 2024; Raffington et al., 2021). For example, a recent study (Hogan et al., 2024) demonstrated that ELA characterized by home threat (e.g., physical or emotional abuse) or community threat (e.g., living in a neighborhood with a higher rate of violent crimes) predicted increased epigenetic age acceleration across adolescence. This increased acceleration, in turn, was associated with higher levels of psychopathology, as evidenced by findings using longitudinal data from the Future of Families and Child Wellbeing Study (FFCWS) (Hogan et al., 2024).

The Importance of Parenting

Parenting behaviors and the quality of parent-child relationships have emerged as significant factors that may moderate the effect of ELA on epigenetic aging trajectories (Brody et

al., 2016; Creasey et al., 2024; Sullivan et al., 2023; Yamaoka & Bard, 2019). Positive parenting practices—characterized by warmth, sensitivity, and responsiveness—are essential in shaping children's developmental experiences and influencing epigenetic regulation (Yamaoka & Bard, 2019). For instance, threat-related adversity has been associated with functional and structural changes in the amygdala (McLaughlin et al., 2019) and accelerated epigenetic aging (Colich et al., 2020). However, nurturing parenting practices have the potential to inhibit or buffer these detrimental effects (Brody et al., 2016; Sullivan et al., 2023). Recent intervention studies have further underscored the impact of parenting on children's epigenetic aging trajectories. Namely, Merrill et al. (2024) demonstrated that children who participated in internet-based parent-child interaction therapy (PCIT)—which promotes positive parenting strategies to manage child behavior—exhibited a slower pace of epigenetic aging compared to those in the control condition. Similarly, Sullivan et al. (2024) found that children involved in child-parent psychotherapy, an evidence-based dyadic psychosocial intervention, showed reduced epigenetic age acceleration. Moreover, several studies have shown that positive parenting increases may buffer adversity's impact on accelerated epigenetic aging (Brody et al., 2016; Creasey et al., 2024; Sullivan et al., 2023)

Current Study

To date, most studies examining the link between ELA and accelerated epigenetic aging have focused on a single developmental stage or concentrated on epigenetic outcomes specifically in adulthood. Additionally, the extant literature has predominantly evaluated the negative impacts of stress or negative parenting behavior on biological embedding, with limited exploration and attention given to protective factors or resilience trajectories. There is a critical need for social epigenetic research that spans multiple developmental stages, integrates multi-

level analyses, and investigates potential protective factors. These approaches may inform more robust resilience models and prevention programs that move beyond deficit-based frameworks and better support youth who have experienced ELA.

To address these gaps, the current study leverages data from the Future of Families and Child Wellbeing Study (FFCWS) to prospectively examine the moderating role of observed positive parenting practices in longitudinal associations between childhood ELA and adolescent epigenetic aging (see Figure 1 for a conceptual model). Building on prior findings linking threat-based ELA to accelerated epigenetic aging (Chang et al., 2024; Del Toro et al., 2024; Hogan et al., 2024), this study focused on threat-based ELA experienced between birth and age 9 occurring in both home (i.e., physical and/or emotional abuse) and community (i.e., crime and/or exposure to violence) settings. We hypothesized that observed positive parenting practices at ages 3, 5, and 9 will buffer the detrimental effects of threat-based ELA on accelerated epigenetic aging in adolescence.

Method

Participants

Publicly available data from the Future of Families and Child Wellbeing Study, an ongoing longitudinal study of 4,898 families from 20 large cities(population 200,000) across the United States (Reichman et al., 2001), was utilized in this study. Families were recruited at the child's birth (1998-2000), and non-marital births were oversampled at a rate of 3:1, resulting in a disproportionate sample of economically disadvantaged families. The present study examined a subset of children (n = 2,039) from the larger sample, who were selected for having complete epigenetic data at age 9 or 15 years. Data for the present study were collected at birth and when

the children were 3, 5, 9, and 15 years of age. See Table 1 for sample demographics. Sex was reported by the mother at birth, and the child's race was self-reported at age 15 years.

Procedure

Data for the present study were collected when the focal child was 3 (2001-2003), 5 (2003-2006), 9 (2007-2010), and 15 (2014-2017) years of age via in-home assessments. Children's saliva samples were also collected during data collection at ages 9 and 15. In all, 86% of children provided saliva samples at age 9, as did 71% of teens at age 15. Data collection and study procedures were overseen by the Princeton University Institutional Review Board.

Measures

Home Threat. A latent variable was created to represent home threat across ages 3, 5, and 9 via the Parent–Child Conflict Tactics Scale (CTS-PC) (Straus et al., 1998). Home threat was defined as exposure to physical and emotional abuse by a primary caregiver. Primary caregivers reported how often physical abuse (e.g., "spanked on the bottom with a bare hand") and emotional abuse (e.g., "shouted, yelled, or screamed") occurred in the past year. Three items for each category were included, and each item was rated on a 7-point Likert scale ranging from "0 - never happened" to "6 - more than 20 times." Higher scores reflect a greater exposure to threats within the home environment. For complete item details and factor analysis results, see Hogan et al (in press).

Community Threat. A latent variable was created to represent community threat across ages 3, 5, and 9, obtained via the National Archive of Criminal Justice Data's Uniform Crime Reports. The data consisted of county-level crime rate data for the location of the focal child's primary caregiver during data collection at ages 3, 5, and 9. The measure of community threat represents exposure to violence in the child's neighborhood, calculated as the sum of violent crime

instances per capita (murder, rape, robbery, and aggravated assault) and property crime instances per capita (burglary, larceny, motor vehicle theft, and arson).

Parenting Practices. Positive Parenting at ages 3, 5, and 9 was measured via the Home Observation for Measurement of the Environment (HOME) Scale by a trained member of the FFCWS team (Bradley & Caldwell, 1984). The HOME Scale captures data regarding the caring environment in which the focal child was raised and has demonstrated strong interrater reliability and internal consistency (Elardo & Bradley, 1981). The observer coded each item on a binary scale of "0 - did not occur" to "1 - did occur." The HOME Scale was previously shown to be associated with prosocial (Blume et al., 2022) and externalizing behavior (Flannery et al., 2023) in the FFCWS. A latent variable factor score for positive parenting across waves was developed using the WLSMV estimator in Mplus, χ^2 (51) = 224.40, RMSEA = .030 [0.026, .034], CFI = .98, SRMR = 0.054, with standardized factor loadings within wave ranging from .68 to .94. Higher scores on the latent positive parenting variable represent higher levels of observed parent praise, positivity, warmth, and encouragement/support across the 3, 5, and 9-year waves.

Biological Markers

Epigenetic Aging. The FFCWS survey subcontractor, Westat Inc. arranged the sample collection. Westat interviewers used the Oragene® DNA Self-Collection kits (OGR-500) (DNA Genotek Inc) to collect child saliva samples during in-home visits for children at age 9 and 15. Available samples [n = 3,945] were assayed using methylation arrays (Infinium Human Methylation 450K and Infinium Methylation EPIC; Illumina) according to the manufacturer's protocol. Samples were excluded if the ENmix R package quality control procedure identified samples as having outlier methylation or bisulfite conversion values or if the sex predicted from the methylation data differed from the recorded sex. Additionally, the cell-type proportion in

each saliva sample was estimated using the Houseman algorithm implemented in the *estimateLC* function in the *ewastools* package, using the children's saliva reference panel (Middleton et al., 2022). Two DNA methylation-based methods were used to estimate epigenetic aging.

DunedinPACE. The DunedinPACE pace of aging (Belsky et al., 2022), previously employed in pediatric saliva samples (Merrill, Hogan, et al., 2024; Raffington et al., 2021, 2023), was examined for epigenetic pace of aging. A value of one in this measure indicates the estimated epigenetic age and chronological age were equivalent, with a greater value indicating epigenetic age acceleration in comparison to chronological age. The development of this DNA methylation biomarker differs from age-focused epigenetic estimators in that it is rooted in the dynamics of health and phenotypes supporting successful biological aging processes (Belsky et al., 2022), including being trained on within-individual decline in 19 indicators of organ-system integrity spanning two decades in the Dunedin Study (Belsky et al., 2022). The FFCWS has two DunedinPACE measures, and the current study used the most updated method (poam45).

PedBE Epigenetic Age Acceleration. Analyses estimating DNA methylation age acceleration in children were conducted using the Pediatric Buccal Epigenetic Clock (PedBE) (McEwen et al., 2020), a clock trained in oral tissue to estimate biological age in children within an error of less than 4 months using 95 sites across the epigenome. PedBE was measured by the residuals of a linear mixed effect model with maximum likelihood estimation of predicted PedBE age on reported chronological age, accounting for predicted buccal epithelial cell proportion (as recommended by the authors of the tool) (McEwen et al., 2020) and a random effect of individual (both year 9 and 15 were included). This was completed in R (4.3.1) with the nlme package. Buccal epithelial cell proportion was estimated using the EpiDISH package and

accounted for during epigenetic age acceleration calculation due to the association of this cell type and age.

Statistical Analysis Plan

A path analysis model was conducted using Mplus, applying Full Information Maximum Likelihood (FIML) for any missing data, and maximum likelihood estimation with robust standard errors (MLR) was used to adjust for possible non-normality. The following fit statistics were employed to evaluate model fit: chi-square, $\chi 2$: $p > 0.05$ excellent, comparative fit index (CFI; > 0.90 acceptable, > 0.95 excellent), root mean square error of approximation (RMSEA; < 0.08 acceptable, < 0.05 excellent), and the standardized root mean square residual (SRMR; < 0.08 acceptable, < 0.05 excellent). Two models were run with a similar set of predictors, with the outcome differing between the two epigenetic aging outcomes. For each model, covariates were child sex, buccal epithelial (BEC) proportion, and age 9 levels of the epigenetic outcome. For each model, the core predictors were home threat, community threat, observed positive parenting, and the two interactions: home threat by positive parenting and community threat by positive parenting. Simple slopes for high, mean, and low levels of positive parenting were estimated and plotted to interpret any significant interaction effect.

Results

Preliminary results

Descriptives for epigenetic aging outcomes across waves suggest the sample, on average, showed no acceleration on PedBE. In contrast, they showed a faster-than-expected aging pace on the DunedinPACE, though there was substantial variability in each outcome (see Table 1). Bivariate correlations are presented in Figure 1. Community, but not home, threat adversity was positively correlated with accelerated epigenetic aging, whereas positive parenting was

negatively correlated with the pace of aging. BEC cell type was correlated with each epigenetic outcome, and child sex was associated with the pace of aging such that girls had a faster pace of aging. Overall, associations support the inclusion of the covariates and proceeding to the primary models.

Primary results

Complete results are detailed in Table 2, while the simplified conceptual moderation model is illustrated in Figure 1. The model fit for the pace of aging model was excellent, with χ^2 (6) = 5.999, p = 0.423, RMSEA = .000 [.000, .029], CFI = 1.0, SRMR = 0.009. Similarly, the model fit for accelerated PedBE epigenetic age was also excellent, χ^2 (6) = 5.815, p = 0.444, RMSEA = .000 [.000, .028], CFI = 1.0, SRMR = 0.008.

For the Dunedin pace of aging outcome, higher levels of childhood community, but not home, threat predicted a faster pace of aging in adolescence over and above the effect of child sex, cell type, and age 9 pace of aging. Further, higher levels of observed positive parenting in childhood predicted a slower pace of epigenetic aging in adolescence. Importantly, the interaction between community threat and positive parenting was significant, while the interaction with home threat was not. For the PedBE accelerated epigenetic aging outcome, higher levels of childhood community and home threat predicted adolescent accelerated epigenetic aging over and above the effect of child sex, cell type, and age 9 PedBE. However, observed positive parenting was not related to PedBE accelerated nor was either interaction significant.

Figure 3 illustrates the simple slopes of the interaction between community threat and positive parenting in childhood on the adolescent pace of epigenetic aging. Results indicate that low levels of observed positive parenting mitigate the influence of community-level adversity,

leading to an accelerated pace of aging regardless of the level of community threat (b = .006, p = .170). In contrast, when positive parenting was average (b = .012, p < .001) or high (b = .018, p < .001), community threat had a longitudinal effect on the pace of aging, with greater threat associated with faster aging. Furthermore, findings suggest that high levels of community threat (b = -.015, p = .354) weaken the effect of positive parenting on pace of aging. In other words, an accelerated pace of aging occurs when either positive parenting is low or community threat is high, regardless of the other factor. Adolescents demonstrated the slowest epigenetic pace of aging when exposed to high levels of positive parenting and low levels of community threat during childhood.

Discussion

Substantial research has highlighted the long-term negative health consequences of growing up in the face of adversity; however, less is known about the biological mechanisms underlying these associations and the protective factors that may mitigate the biological embedding of ELA. This longitudinal study explored the effects of threat-based ELA within the home and community during childhood on accelerated epigenetic aging in adolescence, with a focus on the moderating role of positive parenting. Consistent with prior findings from the FFCWS (Hogan et al., 2024), greater childhood exposure to home and community adversity was associated with accelerated epigenetic aging in adolescence. Importantly, our findings reveal the nuanced interplay between threat-based adversity and supportive parenting in shaping the pace of epigenetic aging. While positive parenting practices—such as praise and encouragement—were hypothesized to buffer the detrimental effects of threat-based adversity, the results suggest a more complex interaction. Specifically, when children experienced *either* low levels of positive parenting *or* high levels of community-based threat, they demonstrated a faster Dunedin pace of

aging in adolescence. Conversely, the slowest pace of aging was observed among adolescents who experienced high levels of positive parenting or low levels of community threat during childhood development. Notably, moderation effects were not significant for home-based threat, and only main effects emerged for PedBE epigenetic age acceleration, highlighting the specificity of these associations to the context of threat and the type of epigenetic outcome.

Regarding community-based threat, findings from the current study revealed that higher positive parenting was associated with a slower pace of aging in the context of low to average community-based threat. This suggests that while supportive parenting may buffer against some negative effects of environmental stress, its protective influence may diminish under conditions of severe community-based threat. This aligns with previous findings indicating that positive parenting effectively mitigates the impact of mild to moderate stressors but has limited efficacy in the face of overwhelming adversity (Mendez et al., 2016). Additionally, Schumacher and colleagues (2001) identified limited family socioeconomic resources as a consistent risk factor for neglect, underscoring the complex interplay between family resources, parenting practices, and environmental stressors (Schumacher et al., 2001).

The current study's findings align with Bronfenbrenner's bioecological model (Bronfenbrenner & Ceci, 1994), which posits that child development is shaped by interactions within multiple nested systems, such as family- and community-level systems. Proximal factors like positive parenting operate at the microsystem level, while community threats represent exosystem influences, and the interplay between these two systems on child biological embedding aligns with the mesosystem. Such scenarios illustrate how severe community-based threat may exceed the protective “micro” capacity of parental buffering. Importantly, protective factors often work in tandem; for instance, when positive parenting is combined with reduced

community-based threat, their mitigating effects on the pace of aging are amplified- as evidence by this study's findings. Further, though not directly examined in the current study, structural racism and systemic causes of disparities represent the macrosystem and influence community violence (Jay, 2023), parenting (Stern et al., 2022), and biological aging (Krieger et al., 2024). This highlights the need for holistic, multi-level interventions targeting family- and community-level contexts as well as systemic factors that cause inequities across these systems.

Moreover, the lack of evidence supporting our hypothesis that positive parenting buffers against an accelerated pace of aging in high to severe community-based threat environments suggests the presence of a critical threshold beyond which the protective effects of positive parenting may no longer be effective. This threshold likely reflects the overwhelming influence of extreme environmental stressors, which may surpass the buffering capacity of even the most supportive parenting practices. Such findings align with the concept of stress proliferation, as described by Pearlin (1999), which posits that high levels of stress can cascade across multiple domains, compounding over time and ultimately overwhelming existing protective mechanisms. In this context, severe community-based threat may create a cumulative burden of stress that not only diminishes the efficacy of positive parenting but also exacerbates biological vulnerabilities, further accelerating the pace of aging.

As for accelerated epigenetic age, both home threat and community threat significantly predicted this outcome; however, positive parenting did not predict PedBE accelerated epigenetic aging, nor did it interact with either type of adversity. While the significant interaction observed for DunedinPACE but not the PedBE epigenetic clock may seem contradictory, these measures capture distinct aspects of biological aging. Moreover, they do not share any DNA methylation CpG sites despite being epigenetic biomarkers of similar constructs (age vs. aging). Notably, the

DunedinPACE score reflects systemic aging processes across multiple biological domains of health, and is more closely tied to age-related health trajectories (Belsky et al., 2022). In contrast, the PedBE clock was explicitly designed to predict chronological age in children through cheek cells. Thus, while both scores pertain to age or aging, they measure distinct epigenetic mechanisms relevant to the broader construct of biological aging.

Interestingly, prior research on epigenetic effects of early childhood positive parenting interventions has shown divergent findings for these two distinct epigenetic aging outcomes. Sullivan et al. (2023) identified only interactive effects of adversity and increased positive parenting on PedBE accelerated epigenetic aging, Alternatively, Merril, Hogan, and colleagues (2024) reported a main effect of increasing positive parenting, independent of adversity level, on slowing the Dunedin pace of aging. Future research should further investigate the multidimensional nature of epigenetic aging, particularly given widespread disagreement about the definition of biological aging (Gladyshev et al., 2024), the most relevant health-related outcomes for benchmarking aging biomarkers (Herzog et al., 2024), and the limited attention on biological aging during childhood and adolescence (Raffington, 2024).

The absence of an interaction between positive parenting and home-based threat, may be due to the unique dynamics of harsh positive parenting. Research has shown that harsh and supportive parenting practices are not mutually exclusive and can exist simultaneously, which may create a complex and potentially contradictory environment for children (Parent et al., 2016). Such co-occurrence may dilute the protective effects of positive parenting, as the stress induced by threat-based parenting practices (e.g., corporal punishment, hostility) could undermine the benefits of warmth and responsiveness. This aligns with broader theories suggesting that the protective capacity of parenting practices can be context-dependent and may

vary based on the intensity, duration, and type of adversity experienced (Conger et al., 2012) Future research is needed to examine how these parenting practices interact dynamically over time and whether certain combinations of supportive and harsh parenting can have differential effects on biological aging outcomes.

Strengths and Limitations

This study has several limitations that should be considered when interpreting findings. First, the initial sample was disproportionally skewed to non-marital births in economically disadvantaged families, predominately from historically minoritized racial or ethnic backgrounds living in urban areas. Thus, results may not represent the larger United States population or other counties, rural neighborhoods, and a wide array of racial/ethnic groups or genetic ancestry backgrounds. However, social epigenomic research has been predominately conducted with White European ancestry samples, and increased research with populations who experience health disparities is needed to better understand and address the drivers of health disparities and inform the development of effective intervention and prevention programs among various underserved populations (Gillman et al., 2024).

Additionally, we examined adversity and parenting during early childhood to better determine temporal precedence when predicting adolescent epigenetic aging outcomes. Although we controlled for epigenetic aging at age 9, no epigenetic data was available prior to that time point, and our models did not account for concurrent experiences of adversity or parenting during adolescence. Consequently, future research that longitudinally assesses adversity, parenting, and epigenetic aging across all developmental stages from childhood to adolescence—or utilizes an experimental design —will be better equipped to establish temporal precedence, understand the dynamic nature of these associations across development, and infer causality

more robustly. Additionally, DNAm in this study was derived from salivary DNA, and epigenetic outcomes were originally developed using other biological sample collection methods (e.g., cheek swabs, blood), future research should examine whether these associations persist across different tissue types.

The study demonstrates several key strengths in its design and statistical implementation that also should be considered. First, the longitudinal design and multi-method assessment (i.e., parent self-reports, geocoded neighborhood data, observed parenting, and biomarkers) spanning multiple four waves from childhood to adolescence strengthens confidence in findings. Further, we explored multiple domains of threat-based ELA, and different findings based on the environmental context may help guide policy changes or intervention development across socioecological levels to ensure maximum impact on reducing the biological embedding of adversity. Lastly, this study explored the role of positive parenting in buffering the impact of adversity, which contributes to the growing, but still limited, literature studying mechanisms of biological resilience (Merrill, Konwar, et al., 2024).

Conclusion

In summary, our study underscores the importance of continued research to unravel the multidimensional pathways linking ELA to accelerated epigenetic aging and to identify specific protective factors, such as parenting practices, that may mitigate this relation. Our findings suggest that either high levels of community-based threat exposure or low levels of positive parenting are sufficient to accelerate biological aging. Conversely, the combination of high levels of positive parenting and low levels of community threat is associated with the slowest pace of epigenetic aging. These results emphasize the need for community- and family-level

interventions that aim to reduce exposure to adversities while strengthening socioecological supports available to families and children during the sensitive early childhood period.

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Table 1.

Sociodemographic and study variable descriptives

Variable		**n**	**%**
Child sex	Male	1025	50.3
	Female	1014	49.7
Race / Ethnicity	Black, non-Hispanic	901	46.7
	Hispanic/Latino	511	26.5
	Multi-racial, non-Hispanic	99	5.1
	Other, non-Hispanic	52	2.7
	White, non-Hispanic	366	19.0
Parent education	No high school diploma	640	31.4
	High school or equivalent	627	30.8
	Some college	537	26.4
	College or graduate degree	231	11.4
Poverty ratio	0-49%	351	17.2
	50-99%	359	17.6
	100-199%	513	25.2
	200-299%	326	16.0
	300%+	490	24.0
	Mean	**SD**	**Range**
PedBE Acceleration 9	0.003	0.65	-2.93 – 2.44
PedBE Acceleration 15	-0.003	0.85	-3.24 – 4.01
DunedinPACE 9	1.22	0.16	0.80 – 1.76
DunedinPACE 15	1.27	0.18	0.78 – 1.91

Table 2.

Primary Path Analysis Model Results.

	b	95% CI	*p*
DV: Dunedin PACE Y15			
Community Threat	0.012	0.006 – 0.017	.000
Home Threat	0.007	-0.004 – 0.019	.214
Positive Parenting	-0.040	-0.061 – -0.018	.000
BEC Y15	0.809	0.772 – 0.846	.000
Dunedin PACE Y 9	0.237	0.206 – 0.267	.000
Child Sex	0.055	0.045 – 0.064	.000
CommunityT * Parenting	0.025	0.001 – 0.048	.039
HomeT * Parenting	0.025	-0.023 – 0.074	.308
DV: PedBE EAA Y15			
Community Threat	0.074	0.040 – 0.109	.000
Home Threat	0.114	0.043 – 0.185	.002
Positive Parenting	-0.036	-0.174 – 0.102	.606
BEC Y15	1.420	1.184 – 1.618	.000
PedBE EAA Y 9	-0.576	-0.636 – -0.515	.000
Child Sex	0.080	0.019 – 0.141	.010
CommunityT * Parenting	-0.089	-0.256 – 0.077	.293
HomeT * Parenting	0.077	-0.248 – 0.402	.642

Note. BEC = buccal epithelial cell proportion; Y9 = year 9; Y15 = year 15; CommunityT = community threat; HomeT = home threat

Figure 1.

Conceptual model of the moderating effect of parenting on the link between childhood threat adversity and adolescent epigenetic age acceleration.

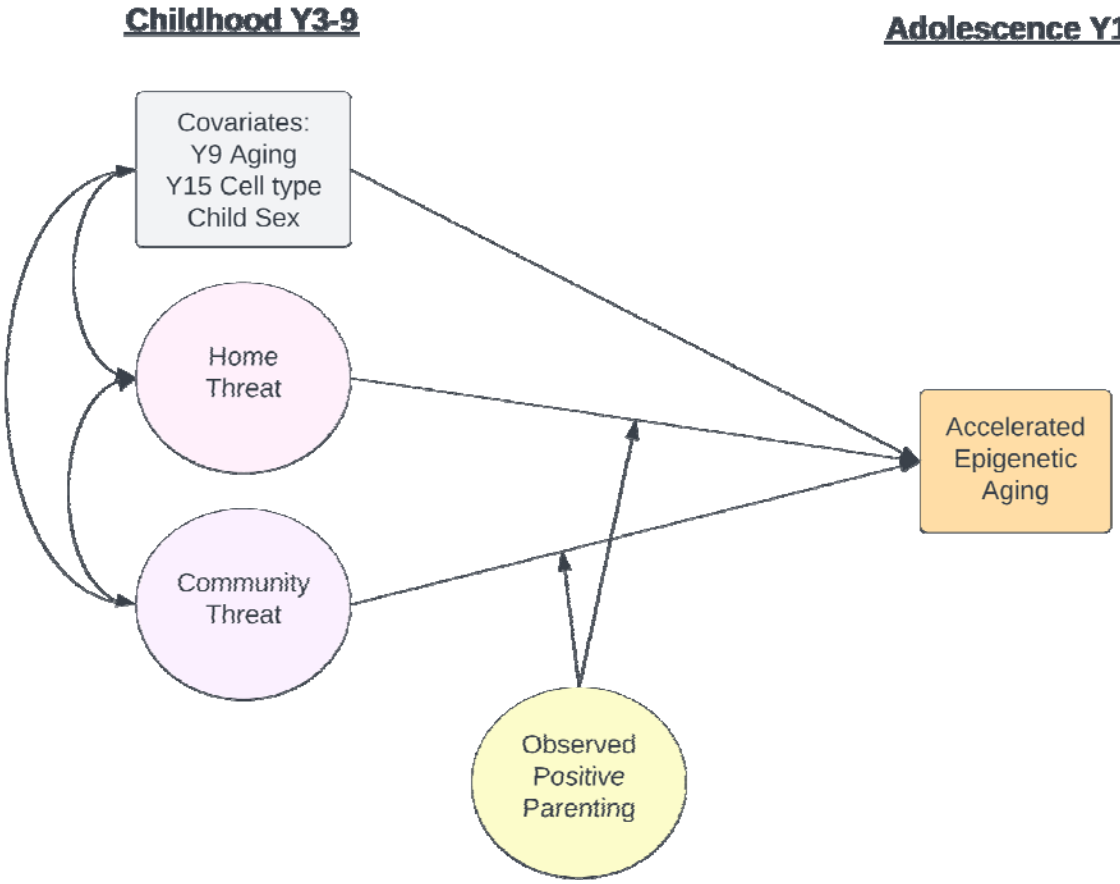
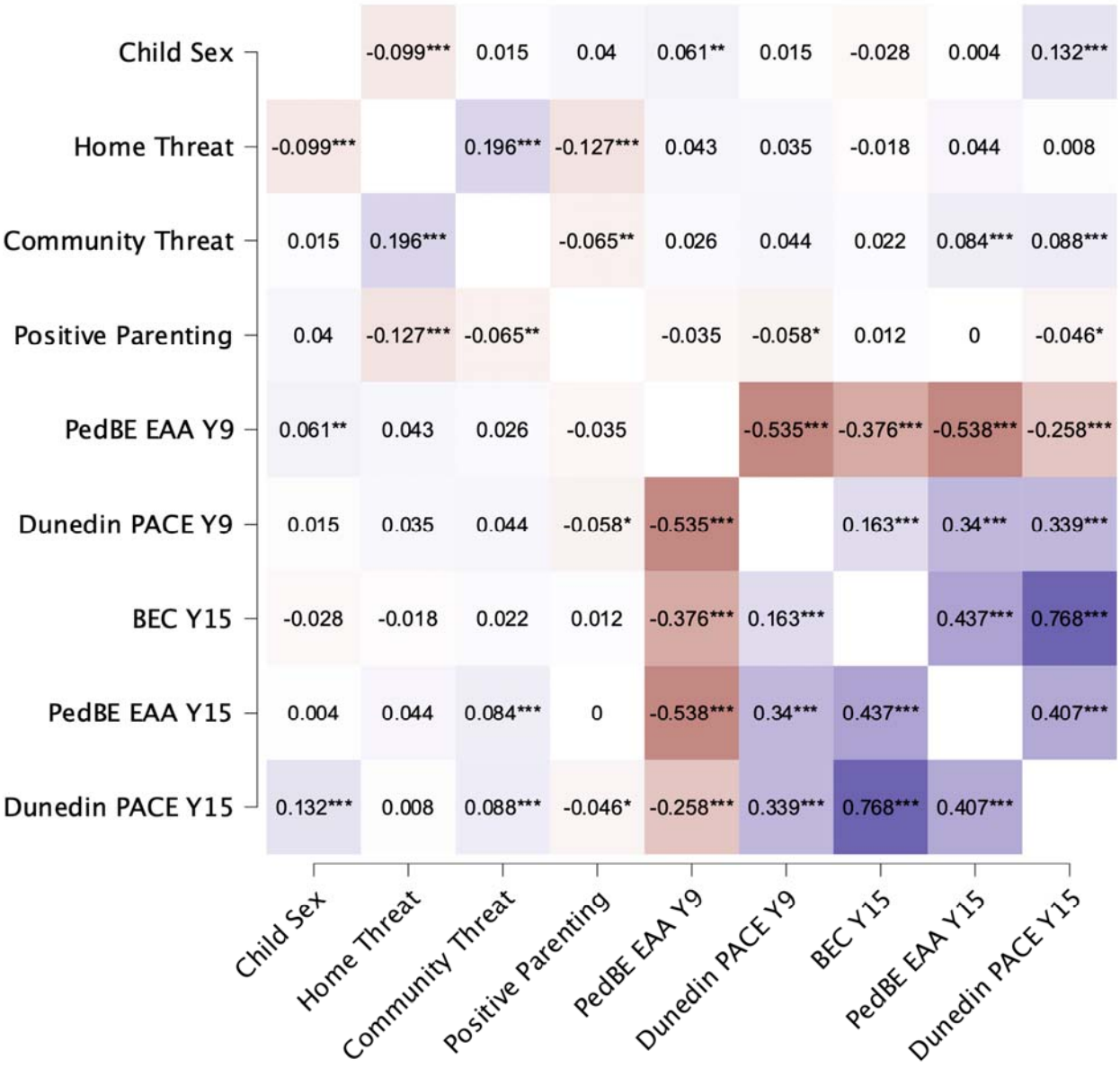


Figure 2

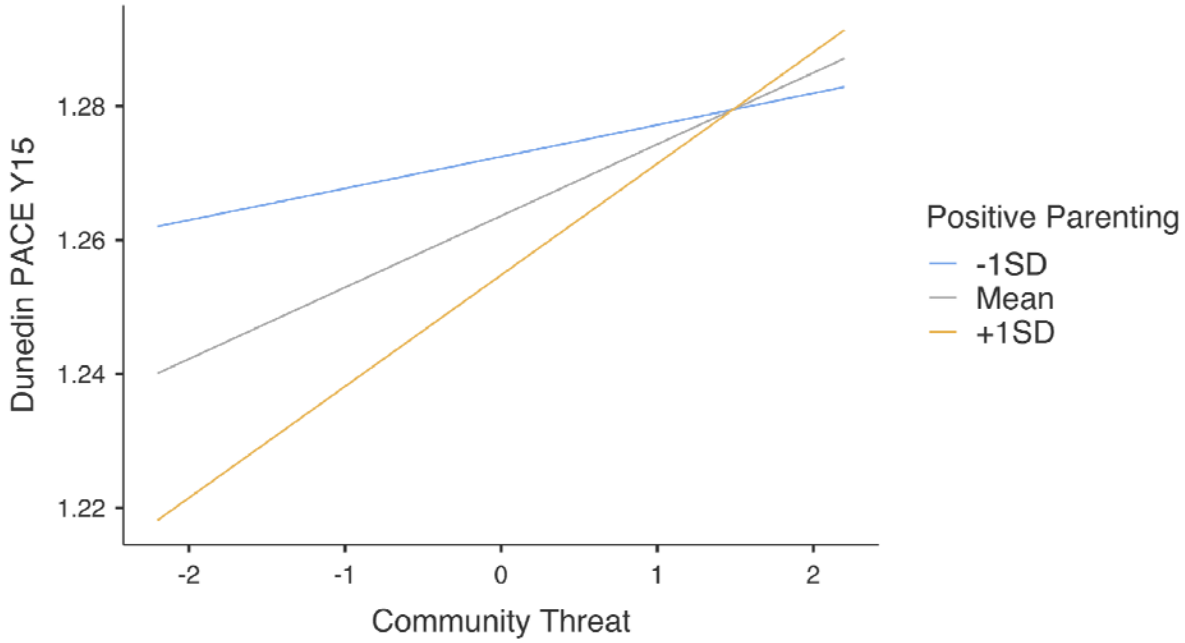
Bivariate correlations heatmap



Note. BEC = buccal epithelial cell proportion; EAA = epigenetic age acceleration; PACE = epigenetic pace of aging.

Figure 3.

Moderation of community threat by positive parenting on epigenetic pace of aging



Note. Plotting points are estimated based on complete data (n = 1904) in Jamovi due to plotting limitations in Mplus